REMARKS/ARGUMENTS

Claims 1-18 are pending in this application. Claim 1 has been amended and claims 2, 4, 5, 12 and 13 have been canceled. No new matter has been introduced as a result of the amendments to the claims.

By the amendments, Applicants do not acquiesce to the propriety of any of the Examiner's rejections and do not disclaim any subject matter to which Applicants are entitled. *Cf. Warner Jenkinson Co. v. Hilton-Davis Chem. Co.*, 41 U.S.P.Q.2d 1865 (U.S. 1997).

Specification

Applicants have corrected typographical errors identified in the specification in paragraphs 0009, 0012, 0019, and 0020 (Table 2). Paragraph 0021 was a duplicate of paragraph 0020 and should have included Table 3 instead of Table 2. Table 3 was disclosed in US patent 6,380,261, which was incorporated by reference in the first paragraph of the instant application. Therefore the correction of paragraph 0021 to include Table 3 does not constitute new matter.

35 U.S.C. §103 Rejections

Claims 1-18 have been rejected under 35 USC §103(a) as being unpatentable over Ornstein (US Pat. 5,527,810) in view of Dreyer (US Pat. 5,597,809).

It is well established that a *prima facie* case of obviousness requires that the Office provide evidence to support three basic criteria: there must be some suggestion or motivation in the cited art to modify a reference or to combine reference teachings. Second, there must be a reasonable expectation of success. Third, the prior art references must teach or suggest all the claim limitations. MPEP 2143.

The Office is respectfully reminded of the case law, namely, that there must be some prior art teaching which would have provided the necessary incentive or motivation for modifying the reference teachings. *In re Laskowski*, 12 U.S.P.Q. 2d 1397, 1399 (Fed. Cir. 1989); *In re Obukowitz*, 27 U.S.P.Q. 2d 1063 (BOPAI 1993) see

also *Takeda Chemical Industries, Ltd. v. Alpharma Pty., Ltd.*, 492 F.3d 1350 (Fed. Cir. 2007).

Also, the Office Action is respectfully reminded that for the 35 USC §103(a) rejection to be proper, both the suggestion of the claimed invention and the expectation of success must be founded in the prior art, and not Applicants' disclosure. *In re Dow*, 5 U.S.P.Q. 2d 1529, 1531 (Fed.Cir. 1988).

Orenstein teaches novel antagonists of NMDA receptors (column 15, lines 30-45) for treatment of a variety of "acute neurological disorders such as cerebral deficits subsequent to cardiac bypass surgery and grafting, stroke, cerebral ischemia, spinal cord trauma, head trauma, perinatal hypoxia, cardiac arrest and hypoglycemic neuronal damage. The formula I compounds are believed to have the ability to treat a variety of chronic neurological disorders such as Alzheimer's disease, Huntington's Chorea, amyotrophic lateral sclerosis, AIDS-induced dementia, ocular damage and retinopathy, and idiopathic and drug induced Parkinson's disease." (emphasis added) The term retinopathy is a general term that refers to some form of non-inflammatory damage to the retina of the eye (Wikipedia). Occular damage is a similarly general term. Neither of these terms provide support for an obviousness rejection for "a disease or condition wherein migration or proliferation of retinal pigment epithelium or glial cells causes or contributes to the cause of said disease or condition" as claimed in instant claim 1. Wikipedia lists the following exemplary types of retinopathies and their putative causes: ocular diseases associated with diabetes (diabetic retinopathy), arterial hypertension (hypertensive retinopathy), prematurity of the newborn (retinopathy of prematurity), sickle cell anemia, direct sunlight exposure (solar retinopathy), medicinal products (drug-related retinopathy), and retinal vein or artery occlusion. Thus, Orenstein uses a general term encompassing numerous species, each representing a unique disease entity having a different etiology. For example, hypertensive retinopathy occurs due to excessively high blood pressure over a prolonged period of time resulting in the small blood vessels that involve the eye becoming damaged, thickened, bulging and leaking. In another non-limiting example, solar retinopathy is damage to the eye's retina, particularly the macula, from prolonged exposure to solar radiation. As discussed

further below regarding the double patenting rejections, different types of retinopathies have different causes resulting in damage or proliferation of different types of cells or tissues resulting in different pathologies and symptoms.

Furthermore, Orenstein does not disclose memantine. Orenstein discloses general formulae with numerous substituent options encompassing hundreds of compounds, none of which is memantine.

Dreyer does not cure the deficiencies of Orenstein. Dreyer discloses treatment of optic neuritis with NMDA receptor antagonists, in one embodiment the NMDA receptor antagonist is memantine. The specification of Dryer lists in columns 2-3 numerous different classes of NMDA antagonists including uncompetitive open channel blocking agents, sigma receptor ligands, competitive NMDA receptor binding agents, agents that are active at the glycine receptor site of the NDMA receptor, and agents that are active at the polyamine site the NMDA receptor, etc. Within these classes Dreyer lists close to 100 compounds that fit within the genus of NMDA antagonists.

The combination of Ornstein and Dreyer do not teach or suggest "a method of treating a disease or condition wherein migration or proliferation of retinal pigment epithelium or glial cells causes or contributes to the cause of the disease or condition, comprising administering a therapeutically effective amount of memantine to the patient suffering from the disease or condition and wherein the disease or condition is not proliferative vitreoretinopathy."

Applicants respectfully submit that Ornstein and Dreyer, in combination, do not teach or suggest each and every element of claims 1, 3, 6-11 and 14-18. Therefore the Office has not established *prima facie* obviousness of claims 1, 3, 6-11 and 14-18 over Ornstein in view of Dreyer.

Double Patenting

Claims 1-6, 8, 9, 11-14, 16 and 17 have been rejected on the grounds of nonstatutory obviousness-type double patenting as being unpatentable over claim 1 of US Pat. 7,230,032 (hereinafter the '032 patent).

Solely in order to advance prosecution of the instant application, and not to concede the propriety of the double patenting rejection, Applicants have amended claim 1 to incorporate the limitations of claim 2 "wherein said disease or condition is not proliferative vitreoretinopathy." However, Applicants disagree with the Office's equating of proliferative vitreoretinopathy in the cited prior art claims with the diseases claimed in the instant application.

The Office has stated on page 4 of the Office Action dated December 28, 2007:

> "Although the conflicting claims are not identical, they are not patentably distinct from each other because they overlap. The claims of the instant application are drawn to the use of memantine for the treatment of proliferative diabetic retinopathy. The claims of the US patent are drawn to the use of memantine for the treatment of proliferative vitreoretinopathy in general. The claims of the instant application are considered to be within the scope of the claim of the US patent."

Proliferative diabetic retinopathy is not the same disease as proliferative vitreoretinopathy and further, proliferative diabetic retinopathy is not a species of the genus proliferative vitreoretinopathy. Additionally, the instant specification (paragraph 00008) and the amended claims specifically disclaim proliferative vitreoretinopathy.

Proliferative vitreoretinopathy refers to the growth of cellular membranes and scar tissue within the vitreous cavity and on the front and back surfaces of the retina (EyeMDLink.com). Diabetic retinopathy comprises two stages, the first of which is a non-proliferative stage in which poor blood sugar control in the diabetic patient leads to overaccumulation of glucose in the small blood vessels of the eye which damages these vessels. The second, proliferative, stage results when lack of oxygen in the retina causes fragile new blood vessels to grow along the retina and in the vitreous cavity

(Wikipedia). Applicants have attached the citations with these definitions in the Appendix.

In summary, proliferative vitreoretinopathy and proliferative diabetic retinopathy are different diseases which involve the growth of scar tissue and cellular membranes within the vitreous cavity and growth of small blood vessels along the retina, respectively. Therefore the claims of the instant application are not within the scope of claim 1 of the '032 patent and not obvious variants thereof that would extend the term of the '032 patent. Applicants respectfully request the withdrawal of this rejection.

Claims 1-6, 8, 9, 11-14, 16 and 17 have been rejected on the grounds of nonstatutory obviousness-type double patenting as being unpatentable over claims 1, 2, 4, 6 and 8 of US Pat. 6,380,261 (hereinafter the '261 patent).

Solely in order to advance prosecution of the instant application, and not to concede the propriety of the double patenting rejection, Applicants have amended claim 1 to incorporate the limitations of claim 2 "wherein said disease or condition is not proliferative vitreoretinopathy." However, Applicants disagree with the Office's equating of proliferative vitreoretinopathy in the cited prior art claims with the diseases claimed in the instant application.

The Office has stated on page 5 of the Office Action dated December 28, 2007:

> "Although the conflicting claims are not identical, they are not patentably distinct from each other because they overlap. The claims of the instant application are drawn to the use of memantine for the treatment of proliferative diabetic retinopathy. The claims of the US patent are drawn to the use of a glutamate receptor agonist, such as memantine for the treatment of proliferative vitreoretinopathy in general. The claims of the instant application are considered to be within the scope of the claim of the US patent."

As discussed *supra*, proliferative diabetic retinopathy is not the same disease as proliferative vitreoretinopathy. Proliferative vitreoretinopathy and proliferative diabetic retinopathy are different diseases which involve the growth of scar tissue and cellular membranes within the vitreous cavity and growth of small blood vessels along the retina, respectively. Therefore the claims of the instant application are not within the

scope of claims 1, 2, 4, 6 and 8 of the '261 patent and are therefore not obvious variants thereof that would extend the patent term of the '261 patent. Applicants respectfully request the withdrawal of this rejection.

Conclusion

Applicants respectfully assert that the pending claims are in condition for allowance and request that a timely Notice of Allowance be issued in this case.

The Commissioner is authorized to charge any fee which may be required in connection with this Amendment to deposit account No. 50-3207.

Respectfully submitted,

Dated: 28 April 2008

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Diabetic retinopathy

From Wikipedia, the free encyclopedia

Diabetic retinopathy is retinopathy (damage to the retina) caused by complications of diabetes mellitus, which could eventually lead to blindness. It is an ocular manifestation of systemic disease which affects up to 80% of all diabetics who have had diabetes for 10 years or more[1]. Despite these intimidating statistics, research indicates that at least 90% of these new cases could be reduced if there was proper and vigilant treatment and monitoring of the eyes.

Diabetic retinopathy *Classification and external resources*	
ICD-10	H36. (http://www.who.int/classifications/apps/icd/icd10online/?gh30.htm+h36) (E10.3 (http://www.who.int/classifications/apps/icd/icd10online/?ge10.htm+e103) E11.3 (http://www.who.int/classifications/apps/icd/icd10online/?ge10.htm+e113) E12.3 (http://www.who.int/classifications/apps/icd/icd10online/?ge10.htm+e123) E13.3 (http://www.who.int/classifications/apps/icd/icd10online/?ge10.htm+e133) E14.3 (http://www.who.int/classifications/apps/icd/icd10online/?ge10.htm+e143))
ICD-9	250.5 (http://www.icd9data.com/getICD9Code.ashx?icd9=250.5)
DiseasesDB	29372 (http://www.diseasesdatabase.com/ddb29372.htm)
MedlinePlus	000494 (http://www.nlm.nih.gov/medlineplus/ency/article/000494.htm) *001212 (http://www.nlm.nih.gov/medlineplus/ency/article/001212.htm)*
eMedicine	oph/414 (http://www.emedicine.com/oph/topic414.htm) oph/415 (http://www.emedicine.com/oph/topic415.htm)

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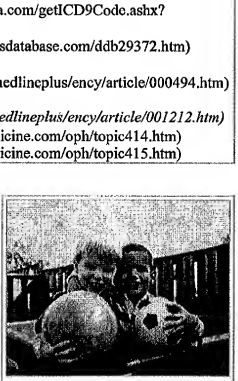

Normal vision. Courtesy NIH National Eye Institute

Signs and symptoms

Diabetic retinopathy often has no early warning signs. Even macular edema, which may cause vision loss more rapidly, may not have any warning signs for some time. In general, however, a person with

macular edema is likely to have blurred vision, making it hard to do things like read or drive. In some cases, the vision will get better or worse during the day.

As new blood vessels form at the back of the eye as a part of *proliferative diabetic retinopathy* (PDR), they can bleed

The same view with diabetic retinopathy.

(haemorrhage) and blur vision. The first time this happens, it may not be very severe. In most cases, it will leave just a few specks of blood, or spots, floating in a person's visual field, though the spots often go away after a few hours.

These spots are often followed within a few days or weeks by a much greater leakage of blood, which blurs vision. In extreme cases, a person will only be able to tell light from dark in that eye. It may take the blood anywhere from a few days to months or even years to clear from the inside of the eye, and in some cases the blood will not clear. These types of large hemorrhages tend to happen more than once, often during sleep.

On fundoscopic exam, a doctor will see cotton wool spots, flame hemorrhages, and dot-blot hemorrhages.

Pathogenesis

Diabetic retinopathy is the result of microvascular retinal changes. Hyperglycemia-induced pericyte death and thickening of the basement membrane lead to incompetence of the vascular walls. These damages change the formation of the blood-retinal barrier and also make the retinal blood vessels become more permeable.[2]

Small blood vessels – such as those in the eye – are especially vulnerable to poor blood sugar control. An overaccumulation of glucose and/or fructose damages the tiny blood vessels in the retina. During the initial stage, called nonproliferative diabetic retinopathy (NPDR), most people do not notice any changes in their vision.

Some people develop a condition called macular edema. It occurs when the damaged blood vessels leak fluid and lipids onto the macula, the part of the retina that lets us see detail. The fluid makes the macula swell, which blurs vision.

As the disease progresses, severe nonproliferative diabetic retinopathy enters an advanced, or proliferative, stage. The lack of oxygen in the retina causes fragile, new, blood vessels to grow along the retina and in

Diabetes mellitus

Types of Diabetes

Diabetes mellitus type 1
Diabetes mellitus type 2
Gestational diabetes

Pre-diabetes:
Impaired fasting glycaemia
Impaired glucose tolerance

Disease Management

Diabetes management:
•Diabetic diet
•Anti-diabetic drugs
•Conventional insulinotherapy
•Intensive insulinotherapy

Other Concerns

Cardiovascular disease

Diabetic comas:
•Diabetic hypoglycemia

the clear, gel-like vitreous humour that fills the inside of the eye. Without timely treatment, these new blood vessels can bleed, cloud vision, and destroy the retina. Fibrovascular proliferation can also cause tractional retinal detachment. The new blood vessels can also grow into the angle of the anterior chamber of the eye and cause neovascular glaucoma. Nonproliferative diabetic retinopathy shows up as cotton wool spots, or microvascular abnormalities or as superficial retinal hemorrhages. Even so, the advanced proliferative diabetic retinopathy (PDR) can remain asymptomatic for a very long time, and so should be monitored closely with regular checkups.

•Diabetic ketoacidosis
•Nonketotic hyperosmolar

Diabetic myonecrosis
Diabetic nephropathy
Diabetic neuropathy
Diabetic retinopathy

Diabetes and pregnancy

Blood tests

Blood sugar
Fructosamine
Glucose tolerance test
Glycosylated hemoglobin

Risk factors

All people with diabetes mellitus are at risk – those with Type I diabetes (*juvenile onset*) and those with Type II diabetes (*adult onset*). The longer a person has diabetes, the higher the risk of developing some ocular problem. Between 40 to 45 percent of Americans diagnosed with diabetes have some stage of diabetic retinopathy. [3] After 20 years of diabetes, nearly all patients with type 1 diabetes and >60% of patients with type 2 diabetes have some degree of retinopathy.[4]

Prior studies had also assumed a clear glycemic threshold between people at high and low risk of diabetic retinopathy.[5] [6] However, it has been shown that the widely accepted WHO and American Diabetes Association diagnostic cutoff for diabetes of a fasting plasma glucose ≥ 7.0 mmol/l (126 mg/dl) does not accurately identify diabetic retinopathy among patients.[7] The cohort study included a multi-ethnic, cross-sectional adult population sample in the US, as well as two cross-sectional adult populations in Australia. For the US-based component of the study, the sensitivity was 34.7% and specificity was 86.6%. For patients at similar risk to those in this study (15.8% had diabetic retinopathy), this leads to a positive predictive value of 32.7% and negative predictive value of 87.6%.

Published rates vary between trials, the proposed explanation being differences in study methods and reporting of prevalence rather than incidence values.[8]

During pregnancy, diabetic retinopathy may also be a problem for women with diabetes. It is recommended that all pregnant women with diabetes have dilated eye examinations each trimester to protect their vision.

Diagnosis

Diabetic retinopathy is detected during an eye examination that includes:

- *Visual acuity test*: This test uses an eye chart to measure how well a person sees at various distances (*i.e.*, visual acuity).
- *Pupil dilation*: The eye care professional places drops into the eye to widen the pupil. This allows him or her to see more of the retina and look for signs of diabetic retinopathy. After the examination, close-up vision may remain blurred for several hours.
- *Ophthalmoscopy*: This is an examination of the retina in which the eye care professional: (1) looks through a device with a special magnifying lens that provides a narrow view of the retina, or (2) wearing a headset with a bright light, looks through a special magnifying glass and gains a wide view of the retina. Note that hand-held ophthalmoscopy is insufficient to rule out significant and

treatable diabetic retinopathy.

- *Ocular Coherence Tomography or OCT*:This is a scan similar to an ultrasound which is used to measure the thickness of the retina. It produces a cross section of the retina and can determine if there is any swelling or leakage.
- *Tonometry*: A standard test that determines the fluid pressure (intraocular pressure) inside the eye. Elevated pressure is a possible sign of glaucoma, another common eye problem in people with diabetes.
- *Digital Retinal Screening Programs*: Systematic programs for the early detection of eye disease including diabetic retinopathy are becoming more common, such as in the UK, where all people with diabetes mellitus are offered retinal screening at least annually. This involves digital image capture and transmission of the images to a digital reading center for evaluation and treatment referral. See Vanderbilt Ophthalmic Imaging Center [1] (http://www.retinopathyscreening.org/) and the English National Screening Programme for Diabetic Retinopathy [2] (http://www.nscretinopathy.org.uk/)
- *Slit Lamp Biomicroscopy Retinal Screening Programs*: Systematic programs for the early detection of diabetic retinopathy using slit-lamp biomicroscopy. These exist either as a standalone scheme or as part of the Digital program (above) where the digital photograph was considered to lack enough clarity for detection and/or diagnosis of any retinal abnormality.

Of the 18 million to 20 million diabetics in the United States, only about half receive annual eye examinations for retinopathy risk. In an effort to increase diabetic patient's compliance for regular eye exams, Digital Healthcare, a Wake Forest, NC company specializing in retinal risk assessment, has announced the introduction of Retasure, a new retinal imaging risk assessment solution that connects primary care physicians with ophthalmic specialists to perform retinal imaging.

Retasure allows primary care physicians to capture digital images of diabetic patients' retinas in a non-invasive procedure that takes just a few minutes. The images are then transmitted over a secure, HIPPA compliant network to a board certified ophthalmologist at an accredited reading center for examination. Results are returned to the primary care physician within 72 hours.

Retasure has been available throughout Europe, and more than one million people have benefited from the system annually.

The eye care professional will look at the retina for early signs of the disease, such as: (1) leaking blood vessels, (2) retinal swelling, such as macular edema, (3) pale, fatty deposits on the retina (exudates) – signs of leaking blood vessels, (4) damaged nerve tissue (neuropathy), and (5) any changes in the blood vessels.

Should the doctor suspect macular edema, he or she may perform a test called fluorescein angiography. In this test, a special dye is injected into the arm. Pictures are then taken as the dye passes through the blood vessels in the retina. This test allows the doctor to find the leaking blood vessels.

Management

There are three major treatments for diabetic retinopathy, which are very effective in reducing vision loss from this disease. In fact, even people with advanced retinopathy have a 90 percent chance of keeping their vision when they get treatment before the retina is severely damaged. Still, the best way of addressing diabetic retinopathy is to monitor it vigilantly and ensure that it does not happen in the first place by careful blood glucose control and limitation of dietary fructose.

These three treatments are laser surgery, injection of triamcinolone into the eye and vitrectomy. It is important to note that although these treatments are very successful, they do not cure diabetic retinopathy. Caution should be exercised in treatment with laser surgery since it causes a loss of retinal tissue. It is often more prudent to inject triamcinolone. In some patients it results in a marked increase of vision, especially if there is an edema of the macula.

Avoiding tobacco use and correction of associated hypertension are important therapeutic measures in the management of diabetic retinopathy. [9]

Laser photocoagulation

Laser photocoagulation can be used in two scenarios for the treatment of diabetic retinopathy.

Panretinal photocoagulation

Panretinal photocoagulation, or PRP (also called scatter laser treatment), is used to treat proliferative diabetic retinopathy (PDR).The goal is to create 1,000 - 2,000 burns in the retina with the hope of reducing the retina's oxygen demand, and hence the possibility of ischemia. In treating advanced diabetic retinopathy, the burns are used to destroy the abnormal blood vessels that form in the retina. This has been shown to reduce the risk of severe vision loss for eyes at risk by 50%. [10]

Before the laser, the ophthalmologist dilates the pupil and applies anesthetic drops to numb the eye. In some cases, the doctor also may numb the area behind the eye to prevent any discomfort. The patient sits facing the laser machine while the doctor holds a special lens to the eye. The physician can use a single spot laser or a pattern scan laser for two dimensional patterns such as squares, rings and arcs. During the procedure, the patient may see flashes of light. These flashes may eventually create an uncomfortable stinging sensation for the patient. After the laser treatment, patients should be advised not to drive for a few hours while the pupils are still dilated. Vision may remain a little blurry for the rest of the day, though there should not be much pain in the eye.

Scatter laser treatment

Rather than focus the light on a single spot, the eye care professional may make hundreds of small laser burns away from the center of the retina, a procedure called *scatter laser treatment* or *panretinal photocoagulation*. The treatment shrinks the abnormal blood vessels. Patients may lose some of their peripheral vision after this surgery, but the procedure saves the rest of the patient's sight. Laser surgery may also slightly reduce colour and night vision.

A person with proliferative retinopathy will always be at risk for new bleeding as well as glaucoma, a complication from the new blood vessels. This means that multiple treatments may be required to protect vision.

Intravitreal Triamcinolone acetonide

Triamcinolone is a long acting steroid preparation. When injected in the vitreous cavity, it results in a decrease in the macular edema (thickening of the retina at the macula) caused due to diabetic maculopathy, along with an increase in the visual acuity. The effect of triamcinolone is transient, lasting

up to three months, and necessitating repeated injections for maintaining the beneficial effect. Complications of intravitreal injection of triamcinolone include cataract, steroid induced glaucoma and endophthalmitis.

Vitrectomy

Instead of laser surgery, some people need an eye operation called a vitrectomy to restore vision. A vitrectomy is performed when there is a lot of blood in the vitreous. It involves removing the cloudy vitreous and replacing it with a saline solution made up of salt and water. Because the vitreous is mostly water, there should be no change between the saline solution and the normal vitreous.

Studies show that people who have a vitrectomy soon after a large hemorrhage are more likely to protect their vision than someone who waits to have the operation. Early vitrectomy is especially effective in people with insulin-dependent diabetes, who may be at greater risk of blindness from a hemorrhage into the eye.

Vitrectomy is often done under local anesthesia. The doctor makes a tiny incision in the sclera, or white of the eye. Next, a small instrument is placed into the eye to remove the vitreous and insert the saline solution into the eye.

Patients may be able to return home soon after the vitrectomy, or may be asked to stay in the hospital overnight. After the operation, the eye will be red and sensitive, and patients usually need to wear an eyepatch for a few days or weeks to protect the eye. Medicated eye drops are also prescribed to protect against infection.

Other treatments

Though not yet commercially available, c-peptide has shown promising results in treatment of diabetic complications incidental to vascular degeneration. Once thought to be a useless byproduct of insulin production, it helps to ameliorate and reverse many symptoms of diabetes[11].

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- Basic ophthalmology for medical students and primary care residents, 7th edition
- Kawasaki T, Ogata N, Akanuma H, Sakai T, Watanabe H, Ichiyanagi K, Yamanouchi T. *Postprandial plasma fructose level is associated with retinopathy in patients with type 2 diabetes.* Metabolism 2004;53:583-8. Fulltext (http://www2.us.elsevierhealth.com/scripts/om.dll/serve?retrieve=/pii/S0026049504000320&). PMID 15131761 (http://www.ncbi.nlm.nih.gov/pubmed/15131761).
- Eye Care for Diabetics (http://www.diabetescaregroup.info/eye-care-for-diabetics/)

External links

- Diabetic Retinopathy (http://www.nei.nih.gov/health/diabetic/retinopathy.asp) Resource Guide from the National Eye Institute (NEI).
- National Diabetes Information Clearinghouse (http://diabetes.niddk.nih.gov/)
- Lions Eye Institute, Perth, Australia (http://www.lei.org.au/)
- Educational website on Diabetic Retinopathy (http://myweb.polyu.edu.hk/~05708076d/nt3/)
- Diabetic Retinopathy - Treatment & Prevention (http://diabetes-abc.blogspot.com/2007/10/diabetic-retinopathy-treatment.html)
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Retrieved from "http://en.wikipedia.org/wiki/Diabetic_retinopathy"
Categories: Ophthalmology | Diabetes | Blindness
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nonprofit charity.

Proliferative Vitreoretinopathy Treatment

Proliferative vitreoretinopathy (PVR) is the most common complication following a **rhegmatogenous retinal detachment**, i.e., a retinal detachment associated with a retinal hole or break. PVR refers to the growth of cellular membranes within the **vitreous** cavity and on the front and back surfaces of the **retina**. These membranes, which are essentially scar tissues, exert traction on the retina and may result in recurrences of retinal detachment, even after an initially successful retinal detachment procedure. PVR may be associated with spontaneous reopening of otherwise successfully treated retinal breaks and may even cause the development of new retinal breaks. Finally, PVR may be associated with severe distortion and "stiffness" of the retina, as a result of the contracting membranes. This aspect of the condition not infrequently results in disappointing visual results, despite the very best of management.

Indications for PVR Surgery

- A confirmed diagnosis of proliferative vitreoretinopathy (PVR)

Surgery for Proliferative Vitreoretinopathy

Surgery for PVR begins with a **vitrectomy**

(pars plana vitrectomy) procedure, in which the vitreous humor is removed. The vitreo-retinal surgeon then performs a **membrane peeling** procedure, in which the contracting membranes on the retinal surface are carefully peeled away from the retinal surface. Finally, a scleral buckling procedure may be utilized in some cases. The surgeon will also typically treat the retina surrounding any retinal tears or holes with laser to help maintain closure of the retinal breaks.

Following the vitrectomy procedure, the surgeon usually instills special gases or fluids into the eye to help flatten the retina and keep it reattached to the outer wall of the eye. If gases are instilled in the eye, head positioning following surgery (for days or weeks) may be necessary to help keep the retina attached. If silicone fluid is placed in the eye to help maintain the retina in the attached position, it must eventually be removed from the eye in the majority of cases.

What to Expect After Surgery for PVR

It is important to realize that recovery of vision after surgery for PVR may take many months. About 50% of patients will regain some useful vision in the affected eye. The level of vision regained, however, is often referred to as "ambulatory vision," indicating vision good enough to see large objects at a close range. The likelihood of regaining vision well enough to read is quite low.

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